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First assessment of population exposure to perfluorinated compounds in Flanders, Belgium

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ABSTRACT

With the objective to evaluate exposure of the population in Flanders (Belgium) to perfluorinated compounds (PFCs), we measured perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) in settled dust in homes and offices, in a selection of food items from local origin, in drinking-water and in human serum. We complemented the data with results from a literature survey. Based on this dataset we calculated intake by children and adults from food, drinking-water, settled dust and soil, and air. Dietary exposure dominated overall intake. For adults, average dietary intake equalled 24.2 (P95 40.9) ng PFOS kg^{-1} d^{-1} and 6.1 (P95 9.6) ng PFOA kg^{-1} d^{-1}, whereas for children the dietary intake was about 3 times higher. Predicted intake is high when compared to assessments in other countries, and to serum levels from Flanders, but comparable to the intakes published by The European Food Safety Authority (EFSA) in 2008. Intake of PFOS and PFOA remained below the Tolerable Daily Intake.

1. Introduction

Perfluorinated compounds (PFCs) are a group of chemicals with a fully fluorinated hydrophobic carbon chain attached to a hydrophilic head. They have been produced since the 1950s and are used in a wide range of applications. PFCs show a high thermal, chemical and biological stability and have bioaccumulating properties. They are found in biota all over the world and in human blood and breast milk (Kannan et al., 2004). Perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) are the most studied PFCs. The majority of PFOS related chemicals are high molecular weight polymers in which PFOS constitutes only a fraction of the total molecular weight (OECD, 2002). PFOS related chemicals have been used in surface treatment, paper protection (food and non-food packaging) and performance chemicals (fire fighting foams, mineral and oil well surfactants, coating additives). PFOA is used to make fluoropolymers that are used for non-stick surfaces (cookware), waterproof breathable membranes for clothing, and in many industry segments for their fire resistance, oil, stain, grease and water repellence. Little information is yet available on the occurrence of PFOS, PFOA and related compounds in environmental compartments and food in Europe and on exposure of the European population. The majority of measurements in food are from fish and seafood (EFSA, 2008), data for other food items are limited and a quantitative assessment is hampered by insufficient analytical sensitivity and performance. Exposure can take place during early life through breast-feeding and at later ages via intake from food and drinking-water, ingestion of settled indoor dust and soil particles and inhalation of air. Dermal transfer has been reported for PFOA (Washburn et al., 2005). Exposure through direct contact with or use of consumer articles cannot be excluded, but seems to be low compared to exposure from food (Washburn et al., 2005; Trudel et al., 2008; Vestergren et al., 2008). Toxicological reference values for PFOS and PFOA were recently published by the European Food Safety Authority (EFSA, 2008).

With the objective to evaluate exposure of the population in Flanders (Belgium) to PFCs, a series of perfluorinated sulfonates and perfluorinated acids was analyzed in settled dust in homes and offices, in selected food items from local origin, in drinking-water and in human serum. The results of these measurements were complemented with literature data and taken forward in an exposure assessment of children and adults to food, drinking-water, settled dust and soil, and air. The exposure assessment was limited to PFOS and PFOA due to the high number of non-detects in food for

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other perfluorinated sulfonates and acids. Intake was compared with serum measurements and with the Tolerable Daily Intake (TDI) values published by EFSA (EFSA, 2008).

2. Methods and materials

2.1. Exposure model

We estimated exposure of the Flemish population to PFOS and PFOA for children (3–<6 yr) and adults. We considered exposure through food and drinking-water, settled dust, outdoor soil, and outdoor and indoor air. We did not consider dermal uptake from contact with dust and soil as – from our experience with contaminated sites – this pathway is generally negligible compared to oral intake from dust and soil. We also did not consider dermal uptake from contact with water as the available data (Washburn et al., 2005) indicate that the skin permeability coefficient is low. Intake rates were either directly expressed per kg body weight and per day or were divided by the average body weight if expressed per day (Table 1). Intake rates were multiplied by concentration to obtain intake per kg body weight and per day. A schematic representation of the modelling framework is given in Fig. S-1 of the Supplemental Information and is explained hereafter.

Intake from food and beverages by adults was calculated using the data from the Belgian Food Consumption Survey 2004 (De Vriese et al., 2006), providing consumption data from 2-d 24 h recalls for individuals of 15 year and older. Consumption data were converted to intake of raw product, and composite food items were converted to their primary ingredients. Multiplication of the consumption data with their corresponding concentration resulted in a short-term intake per kg body weight for each individual and interview day, which was used to calculate the average contribution of food groups to total dietary intake. Long-term intake of the population was estimated from the short-term intake with the NCI method (Tooze et al., 2006), using the Mixtran v1.1 and Distrib v1.1 macros available on the NCI website (http://riskfactor.cancer.gov/diet).

Population average food consumption data for children were taken from the XtraFood model (Seuntjens et al., 2006). The values are based on a food consumption study in 1800 children conducted in 2003, where parents and teachers recorded food and drink consumption by the children for 3 d (3d Estimated Dietary Records) (Huybrechts et al., 2008). Intake was divided by the average body weight for children aged 3–6 year.

Information on time spent outdoor and indoor is needed for the calculation of intake through dust ingestion and through inhalation. Data for the Flemish population aged 12 yr and older were extracted from the Belgian Time Budget Study (Glorieux and Minnen, 2008). We distinguished between working and non-working adults and considered time at work as time spent in the office. Time spent outdoor included transportation by bike, motorcycle or motor bike. Time spent indoor included transportation by car, bus, or train. Time spent in transportation was not accounted for in case of dust ingestion. Time-activity patterns for 3–<6 yrs old children were derived from a local study in about 500 children (Standaert et al., 2009).

Intake values for indoor dust and (outdoor) soil were taken from Van Holderbeke et al. (2007). Daily dust ingestion is calculated by multiplication with time spent indoors at home and in the office (the latter only for adults). The estimates for daily intake of outdoor soil are based on tracer studies. As tracer data do not distinguish well between outdoor soil and indoor soil-derived dust, there is a partly overlap with the dust ingestion values.

Intake through inhalation is calculated using the daily inhalation rates of Brochu et al. (2006), expressed on a body weight basis. Intake is calculated using time-weighted air concentrations, taking into account time spent in the outdoor and indoor environment.

Calculations were run for P50 and P95 concentrations in environmental compartments. Average concentrations were used for food as these are considered representative of longer-term exposure conditions in the general population.

Probabilistic exposure calculations were run to evaluate the variability in exposure due to variation in exposure parameters. We accounted for variation in food intake, soil and dust intake and inhalation rate. The variability in adult dietary intake results from the usual intake calculations. For children's dietary intake, we assumed a probability distribution with the same relative standard deviation as for adults. The variability in soil intake, dust intake and inhalation rate was assessed by setting probability distributions on the parameters. The probability distributions for these parameters are given in Table 1. MonteCarlo simulations were then run using the Crystal Ball software. In each MonteCarlo run, a parameter value is drawn at random from its probability distribution and used as input for the calculation. A number of 1000 trials were run, resulting in a probability distribution on the output.

Contribution of oral and inhalation exposure was compared assuming equal absorption by both routes. Absorption from food is about 95%, based on rat studies (Johnson et al., 1979; Van Den Heuvel et al.,1991). Studies in rats indicate that PFOA is well absorbed by the inhalation route (Kennedy et al., 2004; Hinderliter et al., 2006).

Table 1
Exposure factors used for the calculation of exposure to PFOS and PFOA.

	3–<6 Yrs	21–<31 Yrs (adults)
Body weight (kg)	17.6	68.5
Time outdoors – workers (h d^{-1})	Na	0.65
Time working workers (h d^{-1})	Na	4.85
Time in transport workers (h d^{-1})	Na	1.27
Time indoors workers (h d^{-1})	Na	17.23
Time outdoors – non-workers (h d^{-1})	3.27	0.87
Time working non-workers (h d^{-1})	0	0.35
Time in transport non-workers (h d^{-1})	0.5	0.65
Time indoors non-workers (h d^{-1})	20.23	22.13
Soil intake rate (mg d^{-1})	LN(63, 11)	LN(46, 9)
Dust intake rate (mg h^{-1})	LN(2.06, 3.81)	LN(0.48, 0.49)
Inhalation rate (m^3 kg^{-1} d^{-1})	N(0.44, 0.04)	N(0.25, 0.04)

Na: not applicable.
LN: lognormal distribution (average, standard deviation), N: normal distribution (average; standard deviation).

2.2. Prediction of serum concentrations from external dose

We used a simple, one-compartmental pharmacokinetic model (Fromme et al., 2007) to convert predicted adult dietary intake of PFOS and PFOA to serum levels and compare them with available serum data. At steady-state, serum concentration is related to intake through $C = D \times \mathrm{ABS}/(k \times \mathrm{Vd})$ where C is the serum concentration (ng mL^{-1}), D the intake (ng kg^{-1} d^{-1}), k the first order elimination rate (d^{-1}) equal to $0.693/T_{1/2}$, with $T_{1/2}$ the half-life (d), Vd the volume of distribution (mL kg^{-1} d^{-1}), and ABS the fraction absorbed. We derived probability density functions for $T_{1/2}$ and Vd, based on published literature data. Details are given in the Supplemental Information. For $T_{1/2}$ we derived a log-normal distribution from the data of Olsen et al. (2007). For Vd we derived two probability distribution functions due to the divergence in data. A so-called high Vd estimate was based on the data of Trudel et al. (2008), converting their low, intermediate and high estimate into a triangular distribution. A low Vd estimate was derived from the other literature data, assuming a normal distribution. We assumed oral absorption to be 95% (ABS = 0.95), based on literature data for rats giving absorbed fractions of 0.95 (Johnson et al., 1979; Van Den Heuvel et al., 1991).

2.3. Concentrations in food, serum and environmental compartments

2.3.1. Flemish data

2.3.1.1. Sample collection. Based on the Belgian Food Consumption Survey 2004 (De Vriese et al., 2006), relevant food items in the Flemish diet were selected for analysis. Only food items that were commercially grown and produced in Flanders were included. Food was divided into six groups (vegetables, fruit, meat, milk and eggs, fish, tap water and beer), each containing various food items. Vegetables covered potato, chicory, onion, tomato, carrot, lettuce and leek. Fruit covered apple and strawberry. Meat covered beef, pork and chicken. Fruit and vegetables were obtained through the auction or directly from the grower. Meat was obtained from butchers. Eggs were obtained from chicken farms. Milk came from the cooling tank at the farms. To include variability in concentration, three items were collected and pooled per grower and six commercial growers throughout Flanders were included per food item. This procedure was followed for vegetables, fruit and meat. For fish, eels and cod were caught in respectively Belgian rivers and the North Sea; three individuals were pooled for analysis. Six different beer brands and four tap water samples of different water suppliers were analysed. Food samples were collected between January and June 2008 and were analysed raw.

Blood samples for adults (50–65 yrs) from 8 regions were collected between 2002 and 2005. Regions were characterized as urban agglomeration, rural area, fruit production area, area near waste combustion oven, and industrial region. The objective of the biomonitoring campaign was to reveal differences between regions. Analysis was performed on a pooled sample per region (Roosens et al., 2010). In addition, PFOS and PFOA serum concentrations for the period 2008–2009 were available for 200 adults, aged 20–40 year, from the Flemish Human Biomonitoring Campaign 2007–2011 (Den Hond, 2010). The objective of the latter biomonitoring campaign was to derive reference levels for Flanders.

Volunteers for dust sampling were contacted at random throughout Flanders. In total, 43 houses and 10 offices were sampled. Samples were collected between January and June 2008 by vacuuming according to a standardized protocol as described by Harrad et al. (2008). One square meter of carpet was vacuumed for 2 min and in case of bare floors 4 m^2 for 4 min. Sampling details are given in D'Hollander et al. (2010). Samples were sieved (<500 μm) to ensure particle homogeneity prior to extraction.

2.3.1.2. Sample analysis and QA/QC. Plasma/serum (0.5 mL), tap water (1 L) and beer (300 mL) samples were extracted using a procedure based on Taniyasu et al. (2005) and Kärrman et al. (2007), as described in Roosens et al. (2010).

For food and dust samples, the extraction procedure described by Powley et al. (2005) was used with some modifications, as described in D'Hollander et al. (2010). For dust, 0.5 g was used, whereas for food a sample size of 1 g was used.

The analysis was performed using an ACQUITY UPLC coupled to a tandem mass spectrometer (ACQUITY, TQD, Waters, USA) with an electrospray interface operating in negative ionization mode (ES-MS/MS). Separation was performed on an ACQUITY BEH C18 column (2.1 × 50 mm; 1.7 μm, Waters, USA). Analytical details and QA/QC procedures are given in the Supplemental Information.

2.3.2. Literature data

As a large proportion of food on the Flemish market is from foreign origin, the measured data from local production were complemented with information from other studies with relevant origin. Concentrations in soil and in air were not measured in the current study and data were taken from literature or available studies. To limit the influence of time-trends in levels and the impact of the evolution in analytical methods, we limited the literature search to data collected not earlier than 2003. Only data from European origin were evaluated, except for soil for which information is very scarce.

2.3.3. Processing of the data

The limit of quantification (LOQ) was used as lower limit for all concentrations measured in the current study. For literature data, preference was given to the LOQ, if reported. Otherwise, the limit of detection (LOD) was used.

For settled dust, average and percentile values for PFOS and PFOA were calculated after multiplication of the levels below the LOQ by their "df" value. The "df" value corresponds to the fraction of values in the dataset above the limit of quantification.

For air, food and drinking-water, the data showed variable LOQs and LODs. The "df" value approach could therefore not be used and a medium bound approach was followed in which values lower than the LOQ or LOD were taken forward at half that value.

If food data from various sources were combined, weighted statistical parameters were calculated taking into account the number of samples on which reported averages were based.

3. Results

3.1. Concentrations

P50 and P95 concentrations for indoor dust, soil and air with their references are given in Table 2. More detail is given in Table S-3 of the Supplemental Information.

Lisec (2004) reports soil concentrations of PFOS and PFOA in top soil at the 3M site in Antwerp (Flanders) at locations with high concentrations in groundwater. 3M (2008), Davis et al. (2007) and DuPont (2006) also report concentrations of PFOA in contaminated soil. Data at reference locations and at higher depth were below detection limits. Detection limits for PFOA vary between studies (0.17–15 μg kg^{-1} dry matter). We took half of the detection limit from Lisec (2004) for the calculations.

Jahnke et al. (2007a) report a concentration range of 0.4–1.6 pg PFOS m^{-3} and <0.2–2.6 pg PFOA m^{-3} in air for 3 indoor locations in Hamburg (Germany); Barber et al. (2007) report average concentrations of <47.4 pg PFOS m^{-3} and 4.4 pg PFOA m^{-3} based on measurements for four homes in Tromsø (Norway). We used the highest concentration of PFOS reported by Jahnke et al. (2007a) and the average concentration of PFOA reported by Barber et al. (2007). For outdoor air, we used the data of Barber et al. (2007), Dreyer and Ebinghaus (2009), and Jahnke et al. (2007b). For the latter, we only selected the measurements at Bremerhaven (Germany) and Vigo (Portugal) as being representative for Europe.

The concentrations in food combine both project data and data from the literature. The resulting average and concentration ranges are given in Table 2. The results from the own measurements are reported in Table S-4 of the Supplemental Information. Concentrations of PFOS and PFOA in molluscs and crustaceans are based on measurements in species from Portugal and Italy. Although these regions do not contribute significantly to the origin of molluscs and crustaceans in Flanders, no better data were available. The concentrations in food show large variations, sometimes spanning more than three orders of magnitude (e.g. potatoes). This is caused by the variation in LOQs and by the variation in reported levels. Highest levels are seen in fish and seafood.

3.2. Exposure

Results of the exposure calculations are given in Table 3. The data show that exposure is dominated by intake from food, while other exposure pathways contribute only marginally. Fig. 1 shows

Table 2
Concentrations of PFOS and PFOA in environmental compartments and food.

	PFOS				PFOA			
	P50	P95	*N*	Reference	P50	P95	*N*	Reference
Indoor dust (ng g^{-1})								
Homes	0.73	21.7	40	[1]	0.72	11.4	43	[1]
Offices	1.83	6.88	9	[1]	2.88	56.9	10	[1]
Soil (ng g^{-1})	5[a]		1	[2]	7.5		1	[2]
Air (pg m^{-3})								
Indoor air	1.6[b]		3	[3]	4.4[c]		4	[4]
Outdoor air	1.6	46	38	[4], [5], [6]	8.9	552	34	[4], [5], [6]
	Average (range)		*N*	Reference	Average (range)		*N*	Reference
Food and beverages (ng g^{-1})								
Potatoes	6.18 (<0.021–19)		6	[1]	0.67 (<0.57–2.0)		6	[1]
Vegetables	0.60 (<0.0057–10)		36	[1], [7]	0.65 (<0.027–4.1)		36	[1], [7]
Fruits	0.35 (<0.017–0.7)		11	[1], [7]	0.43 (<0.037–1.6)		11	[1], [7]
Eggs	6.86 (<0.12–22)		8	[1], [7]	0.86 (<0.055–5.0)		8	[1], [7]
Milk and dairy products	0.25 (<0.014–0.64)		9	[1], [7]	0.12 (<0.028–0.34)		9	[1], [7]
Cereals and rice	0.052 (<0.069- < 0.12)		3	[1], [7]	0.055 (<0.08- < 0.12)		3	[1], [7]
Pork meat	0.17 (0.045–0.47)		7	[1], [7]	0.055 (<0.053- < 0.12)		7	[1], [7]
Poultry meat	0.63 (0.02–2.1)		5	[1], [7]	0.055 (<0.067–0.06)		5	[1], [7]
other meat	0.055 (0.03–0.06)		7	[1], [7]	0.52 (<0.034–3.3)		7	[1], [7]
Seafish	12.0 (<0.12–62)		28	[1], [7], [8], [9], [10], [11]	0.59 (<0.065–5.4)		27	[1], [7], [8], [10], [11]
Freshwater fish	174 (1.3–551)		26	[1], [10]	0.78 (<0.6–9.13)		26	[1], [10]
Crustaceans, and molluscs	9.86 (0.148–80)		745	[7], [11], [12]	3.34 (<0.029- < 15)		652	[7], [11]
vegetable oil	0.033 (<0.034–<0.099)		2	[7]	0.091 (<0.115- < 0.25)		2	[7]
Drinking-water, coffee and tea	0.005 (0.004–0.01)		4	[1]	0.002 (0.001–0.005)		4	[1]
Beer	0.013 (<0.0013–0.04)		5	[1]	0.006 (<0.0008–0.02)		5	[1]

[1] This study, [2] Lisec (2004), [3] Jahnke et al. (2007a), [4] Barber et al. (2007), [7] Ericson et al. (2008), [8] Corsolini et al. (2008), [9] Haukas et al. (2007), [10] Kallenborn et al. (2004), [11] Nania et al. (2009), [12] Cunha et al. (2005).
[a] Half of the detection limit.
[b] Maximum value of given range.
[c] Reported average.

Table 3
Estimated average and P95 intake of PFOS and PFOA (ng kg^{-1} d^{-1}) by the Flemish population from food and environmental sources (values between brackets represent P95 intake).

	Soil	Dust		Air		Food
PFOS						
Concentration level	Unsp.	P50	P95	P50[a]	P95[a]	Average
3–<6 yr	0.018 (0.024)	0.0008 (0.003)	0.024 (0.082)	0.00061 (0.0008)	0.003 (0.004)	57.1 (96.6)
⩾21 yr working	0.003 (0.0045)	0.0001 (0.0003)	0.0016 (0.0046)	0.0004 (0.00049)	0.0006 (0.0009)	24.2 (40.9)
⩾21 yr non-working	0.003 (0.0045)	0.00008 (0.0002)	0.0021 (0.006)	0.0004 (0.00049)	0.0008 (0.001)	24.2 (40.9)
PFOA						
Concentration level	Unsp.	P50	P95	P50	P95	Average
3–<6 yr	0.027 (0.036)	0.0008 (0.003)	0.012 (0.043)	0.0022 (0.0026)	0.035 (0.04)	20.1 (31.5)
⩾21 yr working	0.005 (0.007)	0.00015 (0.0004)	0.0027 (0.0074)	0.0011 (0.0014)	0.005 (0.006)	6.10 (9.6)
⩾21 yr non-working	0.005 (0.007)	0.00008 (0.0002)	0.0013 (0.0035)	0.0011 (0.0014)	0.006 (0.008)	6.10 (9.6)

Unsp.: unspecified.
[a] P50 and P95 concentrations could only be calculated for the outdoor air concentrations.

that dietary exposure of children to PFOS is dominated by intake from potatoes (48%), followed by fish and seafood, dairy products, eggs and fruit (each contributing for about 10%). In adults, intake is dominated by fish and seafood (57%), followed by potatoes (28%). The intake of PFOA in children results mainly from fruit (30%) and vegetables (20%), with fish and seafood constituting only a small fraction, whereas the exposure of adults results from fish and seafood, potatoes, fruit and vegetables with almost equal contributions of about 20%. Although concentrations of PFOS and PFOA were detected in drinking-water and beer, their contribution to exposure is less than 1%.

The intake from soil seems to be comparable with high dust exposure. But due to the very limited information on soil concentrations we can only make a rough estimate.

At P50 levels in outdoor air, inhalation exposure is dominated by indoor exposure. At P95 levels in outdoor air, outdoor exposure dominates the calculated dose. However, we have only limited information on the concentrations in indoor air and thus cannot reliably estimate the variation in indoor air concentrations.

Average (P10–P90) serum levels in Flanders equalled 73 (37–98) ng PFOS mL^{-1} and 2.3 (1.4–3.4) ng PFOA mL^{-1} for 2002–2005, and 14.8 ng PFOS mL^{-1} (6.5–25.2) and 3.6 (1.7–5.8) ng PFOA mL^{-1} for 2008–2009. When we convert adult dietary intake to serum levels, we obtain serum levels of 227 (125–357) ng PFOS mL^{-1} at low Vd and 16.5 (8.5–27) ng PFOS mL^{-1} at high Vd, and 76 (37–130) ng PFOA mL^{-1} and 3.4 (1.5–5.7) ng PFOA mL^{-1}, again at low and high Vd.

Comparing overall intake of PFOS and PFOA to their respective Tolerable Daily Intake (TDI) values of 150 ng kg^{-1} d^{-1} and 1500 ng kg^{-1} d^{-1} (EFSA, 2008), indicates that intake by children and adults does not exceed the TDI for PFOS. Total intake of PFOA fills in less than 10% of the TDI.

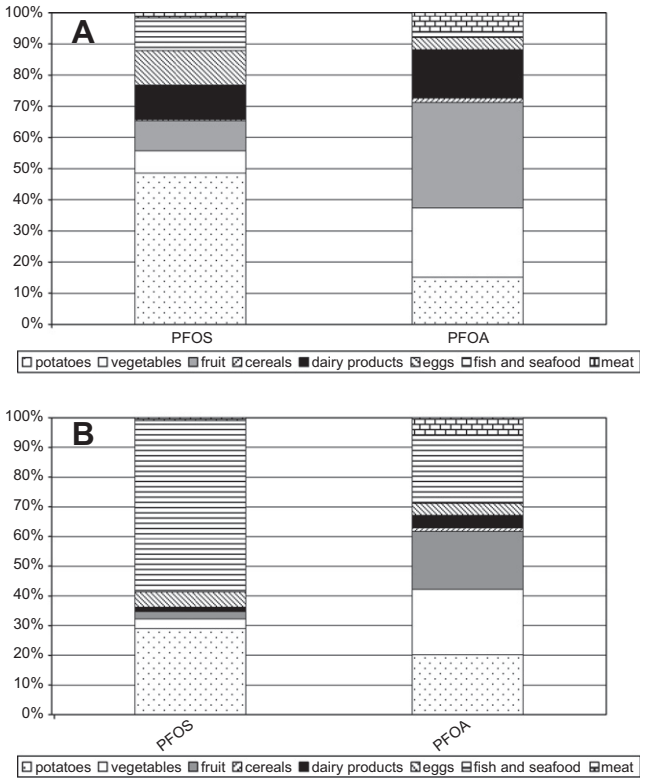

Fig. 1. Contribution of food groups to the dietary intake of PFOS and PFOS in children 3–<6 yrs (A) and adults (B).

4. Discussion and conclusions

An initial exposure assessment of the Flemish population to PFCs in food and the indoor and outdoor environment was realized. However, the exposure assessment still suffers from significant uncertainty. As exposure is dominated by intake from food, the uncertainties therein require further attention. Focusing on commercial products from local origin, the selection of food items was not fully representative for food sold in stores and at markets. Seen the limited number of studies on levels in food items other than fish and seafood, we could not extend our database significantly. Secondly, the high variation of LOQ values, the high number of concentrations below the LOQ and the broad range of reported concentrations hampers an accurate determination of average levels in food. In children, the intake of PFOS is dominated by potatoes, the intake of PFOA is dominated by fruit and vegetables. In adults, fish and seafood become more prominent, but potatoes, fruit and vegetables are still significant contributors to exposure. However, fruit and vegetables have about half of the samples below the LOQ, this being more pronounced for PFOA than for PFOS (see Supplemental Information Table S-4). The measurable concentrations are rather variable and maximum values are even higher than in meat and sea fish. When we perform the dietary intake calculations with lower bound estimates (details not shown), this would reduce the average estimated intake in children by about 12% for PFOS and about 25% for PFOA.

It is thus recommended that further data are collected on the levels of PFOS and PFOA in food on the Belgian market and from local origin and to assess how these levels compare to other countries. This is especially important as one of 3M's production sites is located near to Antwerp, in the north of Flanders and a limited number of samples came from locations within some kilometres of this site. PFOS was produced at the site until the end of 2003, but information on the impact of emissions on the surroundings is not available. The higher levels found here could not explicitly be attributed to samples coming from growers within the vicinity of the 3M plant.

We used the NCI method (Tooze et al., 2006) to extrapolate long-term exposure based on 24 h recall data. This method allows separating the within-individual variation in intake from between-individual variation in intake. The condition to use the NCI method is that the daily exposure distribution approximately shows normality after box–cox transformation (Tooze et al., 2006; Boon et al., 2011). This condition was confirmed by statistical analysis for both PFOS and PFOA. Various methods exist to calculate population intake distributions. Comparison of methods that filter out within-individual variation, which are the preferred ones for calculation of population intakes, shows that they all produce similar results for chemical or nutrient intake calculations (Boon et al., 2011; Souverein et al., 2011).

Table 4 shows the comparison of intake from the diet with assessments from other countries. The dietary intake values separate into two groups, with the one group (FSA, 2006c; EFSA, 2008; Vestergren et al., 2008), to which our study belongs, having estimates which are 10 to 20-fold higher than the estimates in the other group (Fromme et al., 2007; Tittlemier et al., 2007; Ericson et al., 2008). The data of EFSA (2008), where one set of concentrations was used for all countries, show that these differences cannot

Table 4
Average dietary intake of PFOS and PFOA as assessed by various authors (ng kg^{-1} d^{-1}).

	PFOS	PFOA	reference
adults			
Flanders, Belgium	24.2	6.10	this study
United Kingdom / total diet study	10 - 100	1 - 70	FSA (2006)
Germany / duplicate diet	1.8	3.9	Fromme et al. (2007)
Canada / total diet, food of animal origin	1.8	1.1	Tittlemier et al. (2007)
Spain (Catalonia) / market basket	1	-	Ericson et al. (2008)
Italy / fish and seafood, water	58	2.09	EFSA (2008)
the Netherlands / fish and seafood, water	57	2.06	EFSA (2008)
Sweden / fish and seafood, water	45.1	1.69	EFSA (2008)
United Kingdom / fish and seafood, water	49.3	1.82	EFSA (2008)
Western population[a]	18.6	2.64	Vestergren et al. (2008)
children			
Flanders (Belgium) / 3 - < 6 yrs	57.1	20.1	this study
United Kingdom / 4 – 6 yrs / total diet study	50 - 300	4 – 100	FSA (2006)
Spain (Catalonia) / 4 – 9 yrs / market basket	2.35	-	Ericson et al. (2008)
Western population[a] / 1 - 4 yrs	39.6	7.08	Vestergren et al. (2008)
Western population[a] / 5 – 11 yrs	30.0	5.76	Vestergren et al. (2008)

[a] Intermediate scenario; value calculated from reported total absorbed dose by multiplication with food contribution and division by absorbed fraction.

be attributed to differences in food consumption between countries.

The transformation of the dietary intake estimates for adults to serum levels shows an overprediction by a factor 3–10 for PFOS and a factor 30–20 for PFOA, compared to measured serum levels from 2002–2006 and 2008–2009, respectively, when the lower Vd assumption is used. If the higher Vd assumption (based on Trudel et al. 2008) is used, the predicted PFOS and PFOA serum levels correspond well to measured levels. Measured PFOS serum levels from 2002–2005 were higher than from the 2008–2009 campaign, whereas PFOA levels were similar. The apparent decrease for PFOS could be due to the phase-out of PFOS production in Belgium. However, other factors could be influential as the objectives of the two campaigns were different (comparing regions versus determination of reference values, different age groups), the analysis strategy was different (pools per region versus individual samples) and the analytical techniques and labs were different. The finding that the Vd values from Trudel et al. (2008) provides better estimates than the lower Vd values derived by Harada et al. (2003), Butenhoff et al. (2004), Fromme et al. (2007) and Thompson et al. (2010) is not fully in line with similar pharmacokinetic modelling exercises run by Fromme et al. (2007) and Egeghy and Lorber (2011), who suggest that the lower Vd values would be more appropriate. However, there is still substantial uncertainty to this pharmacokinetic model. Pharmacokinetics of PFOS and PFOA are likely better represented by a 2-compartmental model: elimination in cynomolgus monkeys was more rapid at increasing doses (Andersen et al. 2006). Half-life values were derived from studies in occupationally exposed individuals. These are not necessarily representative of the general population. As well, the values used are apparent half-life values and although based on high serum levels compared to those found in the general population, the presence of continuous background exposure (from food and other sources) could lead to increased apparent half-life values. Shorter preliminary half-life values (median of 840 d) were derived by Bartell et al. (2010), who followed PFOA serum levels for half a year in a population exposed through contaminated drinking-water after installation of a water filtration system.

Fromme et al. (2009) used measurements in Canadian home to estimate an average intake from house dust of 31.7 pg PFOS $kg^{-1}\,d^{-1}$ and 16.4 pg PFOA $kg^{-1}\,d^{-1}$ for adults, which is about 20-fold higher than our estimate due to the higher concentrations used. Our estimates for inhalation intake are comparable to those of Fromme et al. (2009) for PFOA, but are lower for PFOS by almost a factor of 10. The difference for PFOS is mainly caused by a difference in input concentrations for indoor air. We used the data from Jahnke et al. (2007a), whereas Fromme et al. (2009) used half of the detection limit reported in Barber et al. (2007).

Taking into consideration all aspects, we conclude that attention should first go to for further refinement of the dietary intake assessment for PFCs, addressing both analytical aspects and representativeness of the food basket. In a second step, this new assessment could then be compared to the available serum levels, taking into account time trends in exposure. Although food dominates intake, there still is a need for reliable data of PFCs in soil, settled indoor dust and (indoor) air.

Finally, there is a need for data on PFCs other than PFOS and PFOA in food and the environment to enable quantitative exposure assessments, as well as for toxicological information to assess the cumulative health risks of these compounds.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.chemosphere.2011.10.034.

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